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Case Report

Frontal Lacunar Infarct in the Development of Non-Convulsive Status Epilepticus: Case Report

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Abstract

Nonconvulsive status epilepticus is characterized by altered mental status without convulsive motor activity and symptoms lasting more than 10 minutes or recorded with electroencephalogram (EEG). This clinical presentation is expected to be seen mostly in chronic epilepsy patients and is recognized especially in critically ill patients, recently. Underlying metabolic anomalies, stroke and infections, may precipitate the nonconvulsive status epilepticus. It is demonstrated in 16% of the patients over 60 years old with altered mental status in the emergency department (ED) and the mortality rates increase by 50% among adults. Ischemic stroke has been shown to be associated with an increased risk of nonconvulsive status epilepticus. In ischemic stroke, not only cortical and great vessel infarcts but also lacunar infarcts have the possibility of developing nonconvulsive status epilepticus. In this case report, we aimed to present with literature information that especially frontal region infarcts may be a determining factor in the development of early status epilepticus after stroke.

Keywords: Frontal, infarct, nonconvulsive status epilepticus

INTRODUCTION

Seizure is a common complication of stroke, and the incidence of post-stroke seizures has been reported to be observed in approximately 7% of all stroke patients.[1] In the studies conducted, the frequency of seizures in the early post-stroke period ranges from 2% to 33%, and 50% to 78% of these occur within the first 24 hours after stroke.[2] Seizure frequency in the late post-stroke period ranges from 3% to 67%.[2,3] In this case, we aimed to present with literature information that especially frontal region infarcts may be a determining factor in the development of early post-stroke period status epilepticus.

CASE PRESENTATION

A 64-year-old, right-handed female patient was evaluated in the emergency service with the complaint of weakness in the distal right wrist. She had a history of hypertension and type 2 diabetes mellitus (DM), and therefore, the patient was using linagliptin, metformin, metoprolol, and

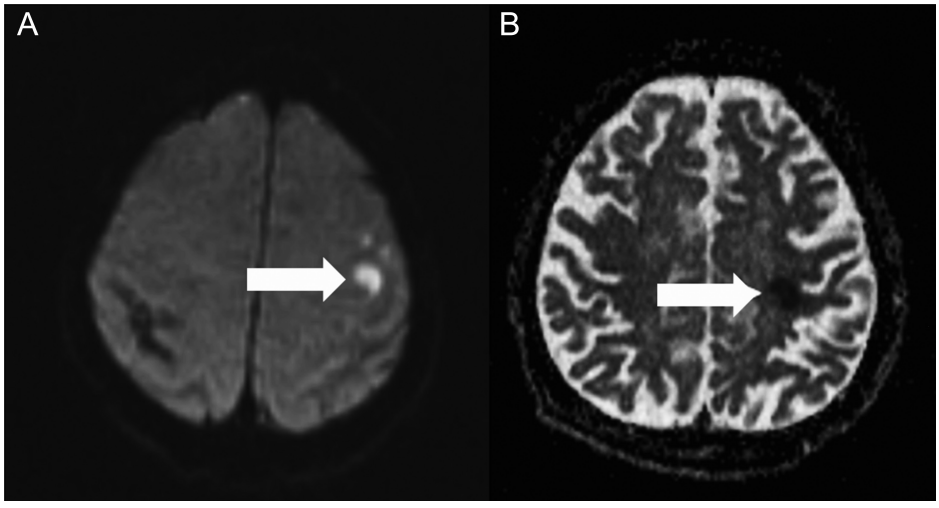

Figure 1 (A, B). Cerebral diffusion-weighted magnetic resonance imaging (DAMRG), A: Hyperintense area at diffusion-weighted sequence left frontal lobe precentral gyrus level, B: Hypointense area at ADC sequence left frontal lobe precentral gyrus level

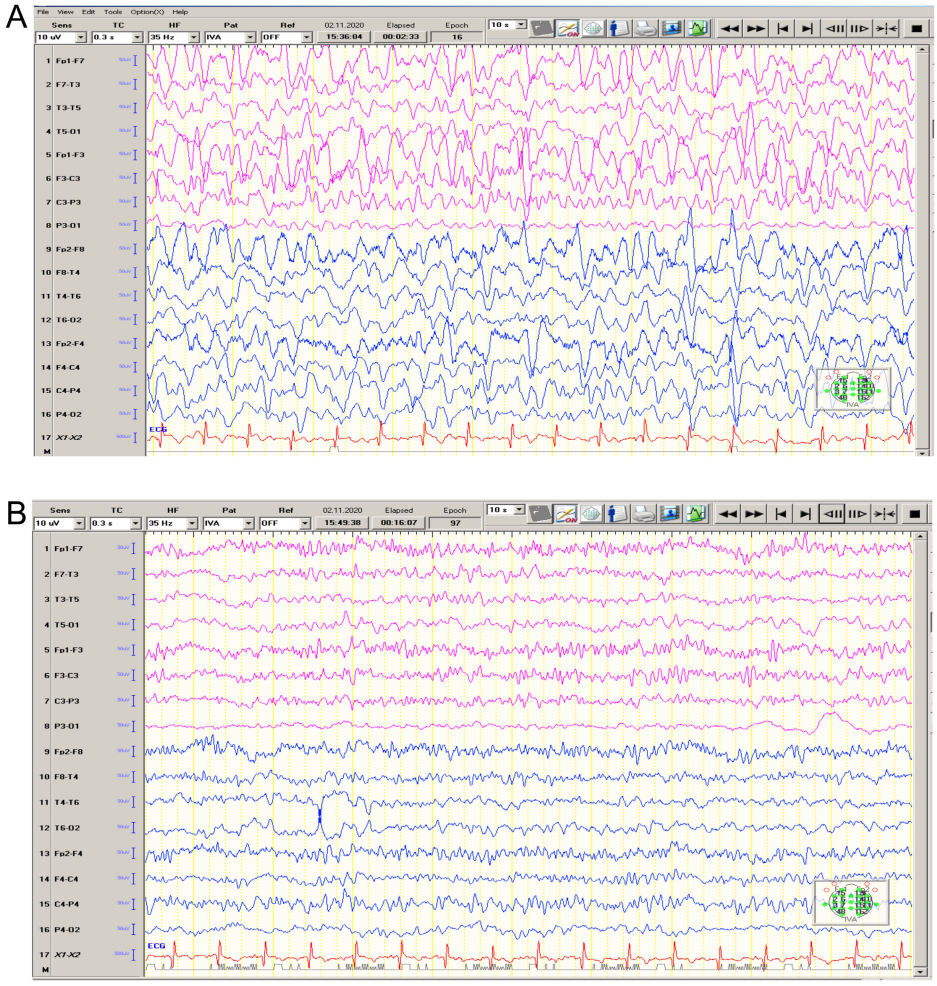

Figure 2 (A, B). A: Bilateral and independent 1.5-2 Hz high-amplitude spike-slow wave and generalized theta-delta waves are observed in the right and left hemispheres.B: EEG suppression after intravenous diazepam.

olmesartan. During the neurological examination performed in the emergency service consultation, the patient was conscious, oriented, and cooperative. Her eye movements were normal and she was able to show direct and indirect light reflexes. During her motor examination, the distal muscle strength of the right wrist was 4/5 while proximal was 5/5. Left upper extremity and bilateral lower extremity muscle strength were also complete. Cerebellar tests of the patient were also normal, and no high cortical dysfunction was detected. Her aphasia examination was evaluated as normal. Her brain computed tomography imaging performed in the emergency service was evaluated as normal. Cerebral diffusion-weighted magnetic resonance imaging (DAMRG) revealed a hyperintense area at left frontal lobe precentral gyrus level in the diffusion-weighted sequence and a hypointense area at left frontal lobe precentral gyrus level in the apparent diffusion coefficient (ADC) sequence. These findings were considered consistent with acute ischemia (Figure 1A and B). The patient was admitted to the neurology service with the diagnosis of acute ischemia, and acetylsalicylic acid 100 mg 1×1 and clopidogrel 75 mg 1×1 treatment was initiated.

MAIN POINTS

- Availability of frontal infarction may be the most important risk factor in the development of post-stroke nonconvulsive status epilepticus (NCSE).
- It should be kept in mind that NCSE may develop although the infarct area is small.
- Nonconvulsive status epilepticus should be considered in the preliminary diagnosis as a cause for loss of consciousness in the early post-stroke period.

On the first day of hospitalization of the patient in service follow-up, levetiracetam (LEV) treatment was initiated due to the presence of focal clonic seizures in the right upper extremity in which awareness was preserved. In the routine EEG, slow wave activity was detected in the left hemisphere at theta frequency with sharp contours. Levetiracetam dose of the patient was gradually increased and a maintenance dose of 2×1000 mg was started. Focal clonic seizures in the right upper extremity completely ceased.

On the third day of the hospitalization follow-up of the patient, routine blood tests and cerebral DAMRG were repeated for the differential diagnosis of the patient's clinical condition, since she developed a tendency to sleep, her gaze became meaningless, and her speech decreased significantly although no seizure was observed in clinical

terms. Routine blood tests were evaluated to be within normal limits, and no additional image suggestive of acute infarction was detected in cerebral DAMRG. Bilateral and independent 1.5-2 Hz high-amplitude spike-slow wave and generalized theta-delta waves were characterized in the right and left hemispheres in the portable EEG (Figure 2A). EEG suppression was observed after intravenous diazepam (Figure 2B). Nonconvulsive status epilepticus (NCSE) was considered in the case with the clinical picture and EEG findings (of the case). Since NCSE may have a structural, infectious, toxic-metabolic, drug-induced, or autoimmune cause, investigations to exclude these causes were evaluated within normal limits.

The diagnosis of drug-induced NCSE has increased remarkably in recent years. Epilepsy or seizures may be observed in patients with DM.[4] Our patient had a history of DM, but her blood glucose values were within normal limits with drug treatment. No case of NCSE was found in the literature regarding the drugs she used. The patient was administered with 3000 mg of LEV in the treatment and her LEV treatment was increased to be 3×1000 mg. Due to the lack of complete improvement in her clinical symptoms and her meaningless gaze, 200 mg of oral lacosamide was administered and a maintenance dose of 2×200 mg was initiated. Her clinical and EEG findings improved completely with dual antiepileptic treatment.

On the tenth day of her hospitalization, since the patient was seizure-free and her clinical course was stable, she was recovered and discharged in a conscious, oriented, and cooperative state with 4/5 muscle strength of the right upper extremity, frust muscle strength of the right lower extremity and mild dysarthric state.

DISCUSSION

Clinical studies point to a difference between early and late post-stroke seizures based on differences in pathophysiology.[5] Early-onset seizures are thought to result from cellular biochemical dysfunction occurring in electrically irritable tissue, while late-onset seizures are thought to result from gliosis and the development of a meningocerebral scar.[6] In addition, stroke severity and cortical involvement are also triggering factors of early seizures that have been consistently identified in studies.[7]

In the literature, time intervals ranging from 24 hours to 30 days have been identified for early post-stroke seizures.[8] Nonconvulsive status epilepticus may persist in 14%-34% of patients with resistant convulsive status epilepticus (CSE). Nonconvulsive status epilepticus after CSE is usually resistant to treatment and has a higher mortality rate. Therefore, early detection and treatment of NCSE is important.[9] Cytotoxic edema and cerebral vasoconstriction during ictal activity cause increased metabolic activity that can lead to cerebral ischemia and increased brain damage. Stroke itself appears to be associated with an increased risk of NCSE. All types of ischemia, that is, not only cortical but also lacunar infarcts, have the possibility to develop NCSE.[10,11]

In a cohort study conducted by Belcastro et al.[12] it was shown that NCSE is not a rare incident following acute ischemic stroke and it occurs in 3%-4% of patients.[12] In our study, significant predictors of NCSE were stated as a large infarct, large artery atherothrombosis, and high National Stroke Health Scale (NIHSS) score during application. In a study conducted by Tomari et al.[13] it was reported that cardioembolic stroke and frontal located stroke may be the most important risk factors for the development of NCSE. In our patient, the infarct area was small and the NIHSS score was not high.

In the treatment of elderly patients with epilepsy, the choice of anti-seizure drug (ASD) is important because of changes in pharmacokinetic parameters, polytherapy, comorbid diseases, and sensitivity to drug effects. In elderly patients with epilepsy, drugs that do not interact with other drugs and do not induce enzyme induction should be preferred as much as possible.[14]

In our case, after electrocardiography was taken, lacosamide was added as the second ASD to the treatment because the cardiac parameters were normal and the loading dose was available.

In conclusion, the presence of an infarct area in the frontal area suggested that it might be the trigger of the seizure observed in the early period. Based on this case, it should be kept in mind that NCSE may develop after resistant CSE and that NCSE may develop in patients we follow up with stroke although the infarct area is small, and the diagnosis should be supported by taking portable EEG at the bedside if necessary.

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